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16. ABSTRACT

This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or q_1^* values are determined for carcinogens for both oral and inhalation routes.

Sufficient data are not available at this time on phenanthrene to determine either acceptable intakes or q_1^*s.

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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with phenanthrene. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) source has been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, OH. EPA 440/5-80-069. NTIS PB 81-117806.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980b) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980b). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q_1*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment evaluation in proper context, refer to the preface of this document. The preface outlines limitations applicable to all documents of this series as well as the appropriate interpretation and use of the quantitative estimates presented.

There are no toxicological data that address effects of phenanthrene by either the oral or the inhalation route. Guidelines concerning PAHs as a class and PAH-containing mixtures specifically address the carcinogenic members of this group. Data are not available which adequately assess the potential carcinogenicity of phenanthrene. Since no data were available, acceptable intakes or carcinogenic potencies could not be estimated.

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LIST OF ABBREVIATIONS

AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
BCF	Bioconcentration factor
CAS	Chemical abstract service
CS	Composite score
GI	Gastrointestinal
PAH	Polycyclic aromatic hydrocarbon
TLV	Threshold limit value
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of phenanthrene (CAS No. 85-01-8) are shown below.

Chemical class:	PAH
Molecular weight:	178.22
Vapor pressure:	6.8×10^{-4} mm Hg at 20°C (U.S. EPA, 1980a)
Water solubility:	1 mg/kg at 25°C (Wise et al., 1981)
Log octanol/water partion coefficient:	4.46 (U.S. EPA, 1980a)
BCF:	1230 (U.S. EPA, 1980a)
Half-lives in water:	9 hours to 2 days (estimated)
Half-lives in soil:	<1 day (estimated)

A quantitative value for the half-life of phenanthrene in the atmosphere could not be located in the available literature. Phenanthrene adsorbed onto particulate matter in the air is expected to be very resistant to both photochemical and chemical reactions (U.S. EPA, 1981). The processes that probably account for significant removal of phenanthrene from the atmosphere are physical removal mechanisms (dry and wet deposition). Although the estimated half-life value for the latter process is unknown, it is expected to be several days. Therefore, phenanthrene may persist in the atmosphere long enough to participate in long distance aerial transport.

The half-life value for phenanthrene in the aquatic media has been estimated from the biotransformation rate constant value (1.6×10^{-7} mℓ $cell^{-1}$ hr^{-1}) given by Mabey et al. (1981) and the concentration range of microorganisms at 10^5 to 5×10^{-5} cell $mℓ^{-1}$ (Burns et al., 1982).

Similarly, the half-life value for phenanthrene in soils has been estimated from the corresponding value in water and the assumption that soils provide much better conditions for biodegradation than aquatic systems (Callahan et al., 1979).

No information pertaining to the mobility of this compound in soil could be located in the literature searched. Based on the soil adsorption coefficient (K_{oc} = 23,000) (Kenaga and Goring, 1980) and the ability to biodegrade in soils, it is unlikely that significant amounts of phenanthrene will leach into groundwater from soils.

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

2.1. ORAL

Specific data regarding GI absorption of phenanthrene are not available, but such data from other structurally related PAHs suggest that phenanthrene is absorbed readily from the GI tract, primarily by passive diffusion (Rees et al., 1971). Also, systemic toxic effects observed following oral administration of related PAHs (Smyth et al., 1962), and the high lipid solubility and ability to cross epithelial membranes of PAH as a class, lend further support to this conclusion (U.S. EPA, 1980a).

2.2. INHALATION

Specific data regarding the pulmonary absorption of phenanthrene are not available, but such data from other structurally related PAHs suggest that phenanthrene is absorbed readily through the lungs (Kotin et al., 1969; Vainio et al., 1976). As a class, PAHs are highly lipid-soluble and can pass across epithelial membranes (U.S. EPA, 1980a).

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. Pertinent data regarding the subchronic oral toxicity of phenanthrene in humans and experimental animals could not be located in the available literature.

3.1.2. Inhalation. Pertinent data regarding the subchronic inhalation toxicity of phenanthrene in humans and experimental animals could not be located in the available literature.

3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic oral toxicity of phenanthrene in humans and experimental animals could not be located in the available literature.

3.2.2. Inhalation. Pertinent data regarding tne chronic inhalation toxicity of phenanthrene in humans and experimental animals could not be located in the available literature.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity of orally administered phenanthrene could not be located in the available literature.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity of inhalation exposure to phenanthrene could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

Pertinent data regarding the toxicant interactions of phenanthrene could not be located in the available literature.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity in humans of oral exposure to phenanthrene could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity in humans of inhalation exposure to phenanthrene could not be located in the available literature.

4.2. BIOASSAYS

4.2.1. Oral. Pertinent data regarding the carcinogenicity in experimental animals of oral exposure to phenanthrene could not be located in the available literature.

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity in experimental animals of inhalation exposure to phenanthrene could not be located in the available literature.

4.3. OTHER RELEVANT DATA

Phenanthrene was negative in the Salmonella typhimurium reverse mutation assay, with or without a metabolic activation system (LaVoie et al., 1981; Oesch et al., 1981). Oesch et al. (1981) noted, however, that positive results could be obtained with phenanthrene in this assay if the amount of the metabolizing enzyme system was increased. Phenanthrene did not induce mutations in cultured mammalian cells, but did increase the incidence of sister-chromatid exchanges in the bone marrow of Chinese hamsters (Oesch et al., 1981).

4.4. WEIGHT OF EVIDENCE

There is no evidence of the carcinogenicity of phenanthrene to humans exposed by the oral or inhalation routes, and this compound has not been tested for carcinogenicity in experimental animals by oral or inhalation exposure. Administration of phenanthrene in three intraperitoneal injections on days 1, 8 and 15 of life at levels of 0.2, 0.4 and 0.8 μmol did not result in an increased incidence of tumors in 100 Swiss-Webster mice (Buening et al., 1979). Phenanthrene is regarded as noncarcinogenic by the U.S. EPA (1980a). IARC (1983) reported that there was insufficient evidence of carcinogenic risk to humans and experimental animals associated with oral or inhalation exposure to phenanthrene. Applying the criteria for evaluating the overall weight of evidence for carcinogenicity in humans proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984), phenanthrene is most appropriately designated a Group D - Not Classified chemical.

5. REGULATORY STANDARDS AND CRITERIA

Exposure criteria and TLVs have been developed for PAHs as a class, as well as for several individual PAHs. The OSHA has set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo[a]pyrene, phenanthrene, acridine, chrysene, pyrene) (Code of Federal Regulations, 1981). NIOSH (1977) recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommends a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

Environmental quality criteria for PAHs, which specify concentration limits intended to protect humans against adverse health effects, have been recommended for ambient water. The U.S. EPA (1980a) has recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic PAHs in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo[a]pyrene, and acknowledges the conservative assumption that all carcinogenic PAHs are equal in potency to benzo[a]pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carcinogenic PAHs over an entire lifetime is estimated to keep the lifetime risk of cancer development below one chance in 100,000.

The U.S. EPA (1980a) has not recommended an ambient water quality criterion for noncarcinogenic PAHs as a class. The EPA acknowledged that data suitable for quantitative risk assessment of noncarcinogenic PAHs are essentially nonexistent.

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. The lack of subchronic oral data precludes the derivation of an oral AIS for phenanthrene.

6.1.2. Inhalation. The lack of subchronic inhalation data precludes the derivation of an inhalation AIS for phenanthrene.

5.2. ACCEPTABLE INTAKE CHRONIC (AIC)

The paucity of data regarding the subchronic and chronic toxicity of phenanthrene precludes the derivation of a CS.

6.2.1. Oral. The lack of chronic and subchronic oral data precludes the derivation of an oral AIC for phenanthrene.

6.2.2. Inhalation. The lack of chronic and subchronic inhalation data precludes the derivation of an inhalation AIC for phenanthrene.

6.3. CARCINOGENIC POTENCY (q_1^*)

6.3.1. Oral. The lack of oral carcinogenicity data precludes the derivation of a unit carcinogenic risk for phenanthrene.

6.3.2. Inhalation. The lack of inhalation carcinogenicity data precludes the derivation of a unit carcinogenic risk for phenanthrene.

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APPENDIX

Summary Table for Phenanthrene

	Species	Experimental Dose/Exposure	Effect	Acceptable Intake (AIS or AIC)	Reference
Inhalation					
AIS	ND	ND	ND	ND	ND
AIC	ND	ND	ND	ND	ND
Oral					
AIS	ND	ND	ND	ND	ND
AIC	ND	ND	ND	ND	ND

ND = Not derived